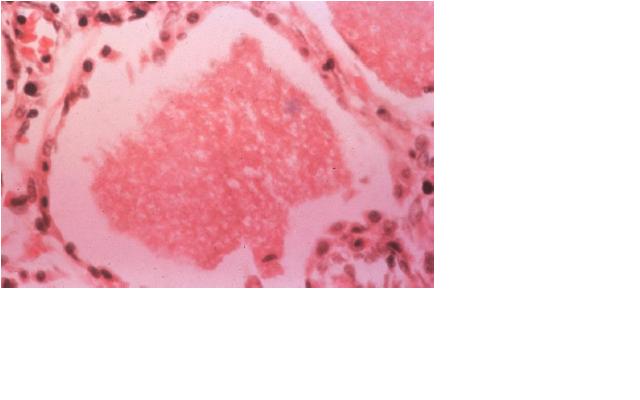
MICRO CASE 22 --- PCP (This is from last week but I made it better for exam time)

A 23 yo man was admitted to the hospital **for fever, non-productive cough, progressive shortness of breath** and fatigue for 2 weeks. He had been diagnosed as **HIV positive** 2 years before, at which time he presented with thrush. He had stopped taking all his HIV-related meds several months ago because of intolerance and he **had progressed to AIDS.**

* PHYSICAL EXAM
  + Thrush present
* LAB STUDIES
  + CD4 T cell count = 80/uL
  + Imaging 🡪 bilateral air space consolidation with **INTERSTITIAL AND ALVEOLAR MARKINGS**
* DIAGNOSTIC WORK UP
  + Cryptococcus neoformans; CMV; H. capitulum; atypical pneumonia; nocardia spp.; PCP; respiratory viruses; TB
  + Rationale: Since HIV, TB and PCP should immediately be considered. CMV usually seen with lower CD T cell counts (<50).
* MICROBIOLOGIC PROPERTIES
  + Fungus (though used to be considered a protozoa)
  + Has a **small trophic form and a 5-8 um cyst from.** 
    - The cyst form has a **thick cell wall** containing **up to 8 intracystic sporozoites**
  + GIEMSA stain used to show the nuclei of trophozoites and intracystic stages while SILVER STAIN used for cyst walls.
* EPIDEMIOLOGY
  + Ubiquitous in nature. By age 5, most children have Abs against PCP. **Disease develops as a result of REACTIVATION of latent infection** in individuals who are immunocompromised (e.g. in those with HIV whose CD4 <200… AIDS DEFINING)
* PATHOGENESIS
  + FIBRONECTIN AND GLYCOPROTEINS of the fungi bind to Type I pneumocytes. Normally, killed by immune system but if immunocompromised, the **cysts rupture** after activation and multiple **trophozoites are released** and fill the alveoli. In those with AIDS, alveolar macrophages defective cuz HIV alters their mannose-Receptor-mediated binding and phagocytosis. The organisms do NOT invade the lung tissue but remain extracellular. As the organisms continue to propagate, basement mem damage 🡪 messed up alveolar capillary permeability 🡺 increased phospholipase activity and **deficiency of surfactant secretion by type II cells (seen in ARDS), causing ventilation/perfusion mismatch and pneumocystis pneumonia**
  + **EOSINOPHILIC Foamy exudate** develops in the alveoli and INTERSTITIAL pathology (**BILATERAL GROUND GLASS APPEARANCE)**



* TREATMENT
  + Bactrim (TMX-SMX) = DOC (tx for 21 days)
  + Alternatives: Bactrim + dapsone, IV pentamidine or atovaquone
  + Steroids used if pO2 <70
* PREVENTION
  + No vaccine
  + Prophylaxis: Bactrim, dapsone, or aerosolized pentamidine
  + Get CD4 T cells > 200